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THE EFFECT OF SMOKING TOBACCO ON THE CARDIOVASCULAR SYSTEM

PART I

During the last century the effects of long-continued use of tobacco on the heart and peripheral blood vessels of man have been investigated widely. The clinical, physiologic and experimental data have been concerned with such problems as the etiologic relationship of the smoking of tobacco to organic cardiovascular disease, the type and degree of change that occur in the cardiovascular system as a result of smoking and whether the mechanical act of smoking, the cigaret paper, the tobacco smoke or nicotine produces these changes.

The use of tobacco has long been suspected of being an etiologic factor in cardiovascular disease and different types of investigations have been carried out to prove or disprove this relationship.

HEART

Various clinical surveys have been made to determine the incidence of smoking of tobacco among patients with angina pectoris. Johnson[1] reported that 70 per cent of sixty patients who had fatal angina pectoris were smokers. A few years later White and Sharber[2] found that in a group of 1,500 patients only 24.4 per cent of the patients with angina pectoris used tobacco to excess in contrast to 33.5 per cent of the patients without angina pectoris who smoked excessively. However, Glendy, Levine and White[3] did find that 93 per cent of their group of eighty-eight men less than forty years of age who were suffering from coronary disease were smokers. Likewise English and Willius[4] who examined 1,000 clinical records found that a greater incidence of coronary disease occurred among smokers than among nonsmokers in the younger age group, that is, among patients less than fifty years of age and perhaps also among those in the age group of fifty to fifty-nine years. Willius[5] in 1942 further stated that it was clear that the incidence of coronary disease increased with the degree of smoking.

Experimental efforts to produce arteriosclerotic changes in the heart and aorta by subjecting animals to tobacco smoke for long periods of time or by subcutaneous or intravenous injections of solutions of tobacco for a considerable length of time have not proved successful.

As early as 1914 investigators using the electrocardiogram studied the effect of toxic doses of nicotine on the hearts of dogs and rabbits and observed nodal rhythm, auricular flutter and heart block. When similar studies were made on normal men an increase in the heart rate and a slight lowering of the T waves only were encountered. However, Neuhof[6] in 1916 reported a case in which sinoauricular heart block was the result of overindulgence in tobacco. More recently Barrow and Ouer[7] in 1943 observed the electrocardiograms of 100 normal men before and after vigorous exercise; three-fifths of these men were smokers. Changes in the P wave in lead CF and in the T waves in the standard leads were found more frequently among the smokers than among the nonsmokers. Graybiel, Starr and White[8] found during the inhalation of tobacco smoke an increase in the blood pressure and heart rate in 80 per cent of forty-five persons of varying ages, some of whom had heart disease. They also observed lowering or inversion of the T waves in the electrocardiogram, which was similar to that observed with atropinization. They attributed this change to the action of nicotine on the cardiac ganglia.

Before and since the use of the electrocardiogram many papers have been written on tobacco heart or tobacco angina. There is still some diversity of opinion as to whether this is a definite clinical entity or whether it is the effect of smoking on patients who already have coronary heart disease. The first prerequisite for such a diagnosis is that smoking must be excessive and must have continued for a long period. In 1915 Allbutt[9] who reported three cases distinguished tobacco angina pectoris from true angina pectoris by the excessive violence and length of the attacks. Moschcowitz[10] in 1928 confirmed this observation and made further suggestions concerning the differential diagnosis as follows: The pains of tobacco angina are more likely to arise during complete rest and may even wake the patient from a sound sleep in contrast to those of true angina which are induced by exertion especially after a meal or by emotion. In some instances the absence of all change in facial color, the absence of physical signs or changes in the pulse rate during the attack, the lack of myocardial change as shown by the electrocardiogram and the freedom from attacks with cessation of smoking further help to distinguish this condition from true angina pectoris. He as well as Aikman[11] and others has noted an apparent sensitization which was characterized by the development of slight pain when the patient was in an atmosphere of tobacco smoke. He expressed the opinion that there are two varieties of tobacco heart (1) that in which evidence of cardiac injury is not present and (2) that in which the signs are suggestive of the intraventricular conduction disturbance associated with coronary and aortic disease. He also pointed out that relief of attacks may not occur immediately after cessation of smoking. Golston[12] in 1937 reported the cases of two excessive smokers. One was an example of unifocal irritation; on cessation of smoking the patient improved in general health and was no longer heart conscious. The second one was an example of multifocal irritation in which onset and remission were sudden. The only treatment was removal of tobacco. As a result of the improvement in these two cases he agreed with Cornwall[13] and suggested that tobacco heart appeared to be mostly a matter of symptoms and functional disturbance. Birk and Huber[14] in 1939 cited three cases (one that of a woman) in which they felt that angina pectoris was caused by excessive smoking. Before the cessation of smoking the electrocardiograms were defi-

nitely abnormal but reverted to normal after cessation of smoking.

White[15] stated that in a few cases of coronary heart disease the use of tobacco has been known to precipitate or to aggravate angina pectoris and to cause tachycardia, temporary changes in the T waves of the electrocardiogram and a harmful effect on the coronary circulation. However, in one healthy young man the inhalation of tobacco smoke has been observed to cause temporary dizziness and inversion of the T waves in leads I and II of the electrocardiogram so that they resembled for a few beats the T waves of coronary heart disease. Whether this change is to be attributed to excessive stimulation of the sympathetic nerves, to coronary arterial constriction or to direct toxic myocardial effect, he and his colleagues do not know but they were inclined to accept the first explanation. Thus White expressed the belief that there is after all such a condition as "tobacco heart" but it is a state of functional derangement of the heart and not an organic heart disease.

In an attempt to determine the mechanism by which the smoking of cigarets produces angina pectoris Ralli and Oppenheimer[16] measured the peripheral circulation by means of the plethysmograph during smoking. They found that in patients who were predisposed to angina pectoris smoking induced peripheral vasoconstriction and at the same time elevation of blood pressure occurred which preceded the onset of precordial pain. They, therefore, suggested that generalized vasoconstriction was present in which the coronary arteries participated. In contrast Graybiel, Starr and White suggested that when attacks of angina pectoris were precipitated by smoking such attacks were not due to coronary vasoconstriction but rather to a sudden increase in the work of the heart as shown by the increase in blood pressure or heart rate or both.

It is interesting to note that previous to these observations Langley and his associates[17] found that an increase or decrease of the blood pressure alone did not cause changes in the electrocardiograms of animals. They increased the blood pressure by clamping the aorta immediately below the diaphragm and the decrease of the blood pressure was accomplished by increasing the intrathoracic pressure by insufflation. Electrocardiograms, taken before, during and after these measures, were unchanged.

HYPERSENSITIVITY

Much investigative work has been done to establish the fact that hypersensitivity to tobacco exists in cases of thrombo-angiitis obliterans and to a lesser extent in cases of coronary heart disease. Harkavy[18] and Sulzberger[19] obtained an allergic skin reaction to nicotine-free tobacco extract in approximately 80 per cent of their group of cases of thrombo-angiitis obliterans and in approximately 40 per cent of their group of cases of coronary heart disease. Sulzberger has suggested that since certain allergens involve certain tissues and even that tissue in certain definite areas only, tobacco may affect the vascular apparatus. Green[20] also has given some support to this theory. In contrast Maddock, Malcolm and Coller[21] could not demonstrate a consistent difference in the responses of cutaneous temperature of the extremities, blood pressure and pulse rate to smoking of persons who showed skin sensitivity to tobacco from those who did not. They also demonstrated that smoking had the same effect on women as on men. This was of particular interest in view of the fact that thrombo-angiitis obliterans is observed infrequently among women and that disease of the coronary arteries is less common in women than in men, tends to appear considerably later in life and is uncommon in the absence of hypertension. Trasoff, Blumstein and Marks[22] found that the incidence of positive skin tests to tobacco extracts is greater among patients who are known to have some allergic condition than among patients who have thrombo-angiitis obliterans and coronary heart disaese. Westcott and Wright[23] also confirmed these findings. Although thrombo-angiitis obliterans and to a less degree coronary heart disease seem to have a statistical relationship to the smoking of tobacco, experimental and clinical evidence of these and other studies indicates that smoking of tobacco most likely is only a contributory factor and not an exciting one.

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